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Cerebral Concussion

by

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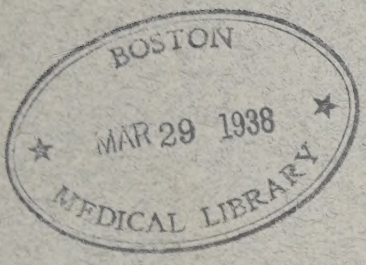

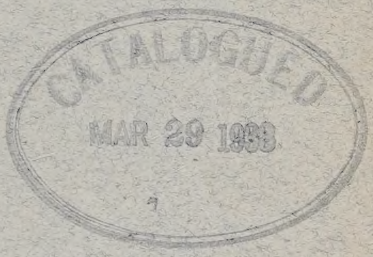

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CEREBRAL CONCUSSION *

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MONTREAL

The present investigation was begun at the suggestion of Dr. Edward Archibald, in order to determine the physiologic response of the brain to an injury which might conceivably cause coincident concussion and compression. The symptoms of concussion, lasting for some hours, might be masked by and have to be distinguished from, those of an oncoming compression. This idea was suggested by clinical observation of certain patients suffering from severe cranial injury, in whom it was found that death supervened with a blood pressure, a pulse rate and a respiratory rhythm which remained approximately normal until near the end. The hypothesis which, in his conception, might explain such unusual behavior on the part of the vital centers, consisted in the assumption that the well-known opposite effects of concussion and of compression were neutralizing each other; that the raised blood pressure, slowed pulse rate and deepened respiration of compression were brought approximately to normal by the antagonistic concussion effects of low blood pressure, rapid pulse rate and hastened, shallow breathing.

In the original few experiments of this research I aimed at establishing, first of all, the effects of pure concussion in the dog and in the cat as a necessary preliminary to later experiments in which I contemplated the addition of more or less simultaneous compression lesions. This was the more necessary in that there admittedly exists, in spite of a considerable body of experimental work, a great deal of indefiniteness, not to say confusion, in the actual knowledge of concussion, particularly as to its physiology and pathology.

It may be said at the outset that these latter experiments designed to elucidate the original hypothesis have been postponed for the time being, because it soon became obvious that our ideas of concussion itself must be more definite, and there must be a much more general agreement than at present exists. Consequently, the work here recorded represents an attempt at establishing the essential pathologic and physiologic foundation of pure concussion.

Concussion is a clinical term applied to a condition following injury of the head. Originally it was broad in its scope, so broad that Galen [1]

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* From the Department of Physiology and Experimental Medicine, McGill University.

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1. Galen, quoted by Polis, A.: Rev. de chir. **14**:273, 1894.

and Hippocrates [2] and later Ambroise Paré [3] included under the name "commotio cerebri," or concussion, all injuries of the head, even severe cerebral laceration and intracranial hemorrhages. With increasing knowledge, there has been a tendency to limit the term concussion to those cases of injury of the head which do not show the clinical syndrome of cerebral pressure, and to those in which contusion and laceration are not apparent. A fine differentiation between concussion and microscopic contusion is not possible at present because there is no general agreement in the literature as to the exact definition of concussion. It may even be said that the literature is contradictory and confusing. Clinical and experimental investigations have proceeded without beginning on a basis of an exact definition of the meaning of the term concussion or of its scope, and so the literature continues to show many divergent views. It is, therefore, primarily necessary to obtain general agreement as to the exact definition of concussion. A pathologic definition hitherto widely taught is that concussion is part of a condition which has for its pathologic basis scattered microscopic contusions and petechial hemorrhages. On the other hand, the word concussion was originally a purely clinical term. With advancing knowledge, especially knowledge of pathology, and owing to the fact that most cranial injuries ending in death showed certain pathologic changes in the brain, the pathologic definition of concussion just indicated naturally became more acceptable. This, however, was at the expense of physiologic argument, and, as I think, unjustly so; for the essential symptoms of concussion undeniably depend on physiologic disturbances, and it has never been proved that these disturbances are caused by anatomic lesions, such as scattered microscopic contusions. Therefore, a clinical physiologic definition should be accepted. The term concussion should be used to indicate a group of symptoms which are the result of a temporary inhibition of cortical function, with or without stimulation or inhibition of one or more of the medullary centers, but which are not accompanied by pathologic lesions.

The conclusion is widely accepted among surgeons that the essentials of concussion are an immediate loss of consciousness, with or without symptoms suggesting medullary effects, and with a progressive tendency toward recovery; further, that pure concussion, that is, concussion without gross lesion, can cause immediate death. If this clinical definition is accepted, the physiologic phenomena and the symptoms may be correlated by means of animal experimentation. The information obtained from hospital cases is naturally somewhat restricted. In the

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2. Adams, F.: The Genuine Works of Hippocrates, New York, William Wood & Company **1**:254.

3. Paré, A.: Oeuvres complètes d'Ambroise Paré, éd. Malgaigne, Paris, J.-B. Baillière **2**:23, 1840.

patients who die of injury of the head, autopsy practically always shows numerous contusions and petechial hemorrhages in the brain. The majority of investigators have concluded that concussion and contusion are synonymous. Rare cases, however, have been reported in which postmortem examination did not show any gross lesion. Such deaths occur almost instantaneously at the moment of the accident. When death occurs later, autopsy always shows lesions caused by contusion. If the foregoing restricted definition of concussion is accepted, the latter cases must be considered as cases of concussion plus contusion. It is consequently unnecessary to endeavor to analyze the symptoms of a mixed condition of concussion and contusion in a study of concussion. The symptoms of concussion are immediate and generalized; those of contusion, progressive and tending to localization. Such a conception eliminates a great deal of the pathologic and physiologic research which has been made concerning concussion, as this research has been based on an investigation of both concussion and contusion. Nevertheless, I shall review this work, as such a review will show the need of a general agreement concerning the definition, and will also be useful later in showing that the term concussion should be limited to designate the condition which is the immediate result of an injury of the head from which the patient gradually recovers or which results in immediate death. Working on this fundamental conception, I shall offer evidence to explain the etiology of concussion, its relation to the symptoms resulting from the effects on the medullary centers, and the relation of concussion to contusion.

SYMPTOMS OF CONCUSSION

Concussion is always caused by an impact on the head, resulting in immediate unconsciousness. Light blows on the head often cause transient vertigo, grogginess, "sparks before the eyes" and similar symptoms but such a condition is of little clinical interest and appears to be largely the result of a mechanical stimulation applied to the central nervous system by means of the blow. Immediate unconsciousness is generally accepted as the constant symptom in concussion. The symptoms of concussion are mainly referable to the cortex, to the medullary centers and to a certain degree to the proprioceptor system, especially the labyrinth. It is presumably the stimulation of the labyrinth which results in the vertigo following the transient unconsciousness. The cortical symptom is the immediate, generalized paralysis of the psychic areas, while the medullary symptom depends on the severity of the blow, and is either stimulation or inhibition of the bulbar centers, which causes changes in the blood pressure, pulse rate and respiratory rhythm.

Cases of concussion are rarely seen by a trained observer from the moment of onset. Gussenbauer's [4] classic case has not this disadvantage

4. Gussenbauer, quoted by Archibald, E.: Am. Pract. Surg., ed. by J. D. Bryant and H. Buck, New York, William Wood & Company **5**:147, 1908.

and will be taken as a typical example of severe concussion. The following account is taken from Dr. Archibald's monograph on surgical affections of the head.

> Gussenbauer and a friend, while on a tour through the Alps, suffered a fall down the Eiger. The friend was rendered unconscious. Gussenbauer fortunately came to no harm. On rising, he found his friend completely unconscious; the pupils were wide; the corneal and other reflexes were abolished; all the muscles were flaccid; the face was deathly pale; and a chance wound was not bleeding. He was not breathing and looked quite lifeless. Evidently, there was both cardiac and respiratory standstill. After a short while, there returned, spontaneously, signs of life; first the pulse, very weak and slow; then the respiration, shallow and slow; then bleeding from the wound. Later, the reflexes appeared; the man gradually began to hear; half opened his eyes; and returned to a condition of semi-consciousness. After a while he fell into a sleep that lasted twenty-four hours; and it was only then that he acquired full consciousness. A week later, all concussion symptoms had disappeared, save that he had lost all memory of the events immediately preceding the accident.

While this example shows the severe type of concussion, most cases are comparatively mild, and there is almost immediate and complete recovery. Probably the best examples of such mild cases are to be found in the boxing ring; these concussions are caused by what is generally known as "the knock out blow." As a result of a sudden blow, usually on the chin, there occurs a sudden loss of consciousness, with flaccid paralysis. The fighter has ten seconds in which to recover sufficiently to get on to his feet and renew the fight, when he may go on to victory. It is inconceivable that such cases are accompanied by any gross lesion, just as it would appear doubtful that Gussenbauer's case was accompanied by a solution of continuity in the central nervous system sufficient to cause such generalized effects. Unless confined within narrow limits to the silent areas, which even then would not be sufficient to cause unconsciousness, gross lesion would give symptoms prolonged over a considerable period. Neurons destroyed in the brain tissue are not repaired but replaced by fibrous connective tissue. This suggests strongly that sequelae are due to gross lesions. Thus the clinical symptoms suggest the limitation of the word concussion to those cases which do not show any gross lesions. Retrograde amnesia may be an exception, but it is unknown whether this is functional or due to an organic lesion.

As all injuries of the head, whether concussion, contusion or hemorrhage, are caused by violence it is obvious that all three must often be present simultaneously, especially in severe cases. Such cases frequently lead to confusion in diagnosis and treatment. In cases of concussion, however, there is either spontaneous recovery or immediate death, so that this diagnosis can be eliminated in those cases in which the course is protracted. Cerebral pressure gives a typical syndrome, with raised

blood pressure and slowed pulse rate. If this conception is accepted, the differential diagnosis of injury of the head becomes comparatively simple, and the factors concerned in the causation of symptoms can be analyzed.

RELATION OF CONCUSSION TO CONTUSION

A review of the literature dealing with the question of the relationship of concussion and contusion shows a great divergence of opinion. Further experimental evidence is required to settle the question as to whether concussion can occur without accompanying gross lesions. Experimental evidence will be given later to support this view.

Boirel,[5] in 1677, was the first to suggest that some cases of injury of the brain were so transient that accompanying lesions of the brain could be excluded. This opinion found marked support in the observations made at an autopsy performed by Littré [6] on a young man who died instantaneously on throwing himself, head first, against a prison wall. Littré did not find any microscopic contusions nor intracranial hemorrhages. Others, Sabatier,[7] and Boyn,[8] and Mourier [9] gave similar negative reports. The diagnosis of concussion thus became limited to those cases which did not show any gross lesions. A reversal of opinion soon occurred, when more careful microscopic observations on the part of better trained men persistently revealed the finer lesions of contusions. Ferrari,[10] working in Kocher's laboratory, showed that in the flinging movement of the brain in response to severe blows on the head, the soft brain struck the bony covering with sufficient force to break small glass ampules buried in the brain tissue at a depth of 5 mm. Kocher,[11] on this evidence, concluded that concussion and contusion were synonymous. Fano [12] considered the symptoms in injury of the head due to contusion, not to concussion. Alquié [13] wrote "la commotio comme lésion differant essentiellement de la contusion est une invention imaginaire." Pirogoff [14] and Beck [15] always found gross

5. Boirel: Traité des plaies de tête, 1677.

6. Littre Acad. roy. de sc. de Paris, 1705, p. 54; quoted by Polis, A.: Rev. de chir. **14**:274, 1894.

7. Sabatier: Med. Operat. **2**:400; quoted by Miles, A.: Brain **15**:154, 1892.

8. Boyn: Thèses de Paris, no. 4, 1818.

9. Mourier: Thèses de Paris, no. 119, 1834.

10. Ferrari, reported by Kocher, in Nothnagel: Spezielle Pathologie und Therapie. Hirnerschütterung, Vienna, Alfred Hölder, 1901, p. 320.

11. Kocher, T., in Nothnagel: Spezielle Pathologie und Therapie, Hirnerschütterung, Vienna, Alfred Hölder, 1901, p. 267.

12. Fano: Mém. sur la commot. du cerveau, Mém. Soc. de chir. **3**:163, 1853; quoted by Polis, A.: Rev. de chir. **141**:278, 1894.

13. Alquié: Gaz. méd. de Paris, 1865, pp. 226, 254, 314, 382, 396, 463, 500.

14. Pirogoff, quoted by von Bergmann: Deutsche Chirurgie, 1880, p. 296; and by Trendelenburg: Deutsche. med. Wchnschr. **36**:1, 1910. Pirogoff: Grundzüge der allgemeinen Kriegschirurgie, Leipzig, p. 77, 1864.

15. Beck, B.: Virchows Arch. f. path. Anat. **75**:207, 1879.

lesions at autopsy in the brains of animals in which they had produced experimental concussion. Bryant,[16] after a careful study of the autopsy reports of Guy's Hospital over a period of twenty-five years, did not find a single fatal case of injury of the head unaccompanied by contusion, laceration or hemorrhage, and concluded that concussion and contusion were synonymous. Le Gros Clarke[17] reports that he had never witnessed a postmortem examination following speedy death from a blow on the head, "which did not show palpable, physical lesion of the brain." Schmaus,[18] Bickeles[19] and Friedlander[20] made careful histologic examinations of the central nervous tissue in man, and also in animals after experimental concussion had been produced, and constantly found minute contusions and petechial hemorrhages. Hauser[21] reported foci of softening in the brain of a man who died six days after an injury of the head; he suggests that had death occurred on the first day these areas would not have been found, and a diagnosis would have been made of fatal concussion unaccompanied by a gross lesion. Beudinger[22] demonstrated numerous degenerated ganglion cells in the brain of a man who died eighteen hours after an injury of the head. Following their experiments of producing concussion by means of repeated light blows, Koch and Filehne[23] performed autopsies on all their animals. They did not find any gross lesions. Scagliosi,[24] however, repeated these experiments and, on careful microscopic examination, found widespread minute lesions. Many other similar discoveries are reported in the bulky literature dealing with this aspect of concussion, but these will suffice to show the strong reasons for the reversal of opinion formed after the publication of Littré's work.

Nevertheless, the conclusion that concussion and contusion were synonymous did not gain universal acceptance. Von Bergmann[25] states that there are undoubtedly cases of death occurring as a result of pure concussion unaccompanied by any anatomic histologic changes. Cush-

16. Bryant, T.: Lancet **2**:405 and 507, 1888.

17. Clark, Le Gros: Diagnosis of Visceral Lesions, ed. 1, 1870; quoted by Miles, A.: Brain **15**:157, 1892.

18. Schmaus, H.: München. med. Wchnschr. **37**:485, 1890; **46**:75, 1899.

19. Bickeles: Arb.. a. d. Inst. Prof. Obersteiner's, III; quoted by Schmaus, H.: München. med. Wchnschr. **46**:79, 1899.

20. Friedländer, C.: Virchows Arch. f. path. Anat. **75**:24, 1879.

21. Hauser, G.: Deutsches Arch. f. klin. Med. **65**:433, 1900.

22. Beudinger, quoted by Archibald, E.: Am. Pract. Surg., ed. by J. D. Bryant and H. Buck, New York, William Wood & Company **5**:150, 1908.

23. Koch, W., and W. Filehne: Arch. f. klin. Chir. **17**:190, 1874.

24. Scagliosi, G.: Virchows Arch. f. path. Anat. **152**:487, 1898.

25. Von Bergmann: Deutsche Chir. **30**:341, 1880.

ing,[26] Trotter,[27] Hutchison[28] and others make similar statements. Savory,[29] while denying the occurrence of death in these cases without accompanying gross lesions, says that the effects must be due to the shock of the violence rather than to the lesions. Since this question remains in doubt, I will give experimental evidence later to support the original assumption that concussion and contusion are separate and distinct clinical entities, frequently associated, but with very different symptoms and prognosis, except in borderline cases.

HYPOTHESIS RELATING TO THE ETIOLOGY OF CONCUSSION

Many hypotheses have been promulgated in regard to the etiology of concussion, none of which have been universally accepted. Following the work of Boirel and Littré, Petit[30] suggested that concussion was due to the vibrations set up in the skull by the blow and transmitted to the brain. This so-called "vibration theory" found wide acceptance, but was disproved by Alquié who demonstrated that the brain did not vibrate under these conditions but moved as a whole. This fact has been amply confirmed since that time by Cushing,[31] Cannon[32] and others.

Felizet[33] first demonstrated that a blow on the skull caused a diminution of the intracranial volume by dropping a blackened skull filled with paraffin on a hard floor. A large round or oval blackened area was made on the floor and the paraffin showed an area of indentation. The cranium, possessing a considerable degree of elasticity, responds to violence by bending inward. Stromeyer[34] was the first to suggest that this lessening of the volume of the cranial cavity forces out the fluids in the skull, thus leading to cerebral anemia which results in concussion. This hypothesis is still widely accepted. Trotter,[27] in 1925, discussing various hypotheses, says: "We can accept none of these hypotheses as a substitute for that which regards cerebral anaemia

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26. Cushing, H.: Surgery of the Head, in Surgery, ed. by W. W. Keen, Philadelphia, W. B. Saunders Company **3**:182, 1908.

27. Trotter, W.: A. System of Surgery, ed. by C. B. Choyce, London, Cassell & Company **3**:468, 1923.

28. Hutchinson, J.: Illustns. Clin. Surg., quoted by Miles, A.: Brain **15**:154, 1892.

29. Savory, W. S.: St. Barth. Hosp. Rep. **5**:72, 1869.

30. Petit, J. L.: Traité des maladies chirurgicales, Paris **1**:1774; quoted by Polis, A.: Rev. de Chir. **14**:275, 1894.

31. Cushing, H.: Am. J. M. Sc. **124**:375, 1902; **125**:1017, 1903; Bull. Johns Hopkins Hosp. **12**:290, 1901.

32. Cannon, W. B.: Am. J. Physiol. **6**:91, 1901.

33. Félizet: Recherches anat. et expérim. sur les fractures des crânes, Paris, 1873; quoted by Polis: Rev. de chir. **14**:280, 1894.

34. Stromeyer, quoted by König, F.: Eléments de path. chir. spéciale, **1**:66; quoted by Polis, A.: Rev. de chir. **14**:281, 1894.

due to hyperacute compression of the brain as the cause of the symptoms of concussion."

Cannon [32] found further support for this hypothesis in his demonstration that the initial rise of intracranial pressure, due to a blow causing inbending of the skull, was sufficient to prevent blood entering the cranial cavity. It is well known that cerebral anemia leads to paralytic symptoms similar to those of concussion. Hill [35] produced unconsciousness and spasm in animals by ligating the four arteries to the head, confirming the results of Kussmaul and Tenner,[36] who produced immediate loss of consciousness and voluntary movement by ligating the innominate artery and the left subclavian. Convulsions occurred in from 10 to 45 seconds. The unconsciousness and the flaccid paralysis seen in syncopal attacks are usually ascribed to cerebral anemia.

In a thorough experimental investigation of concussion, Polis [37] found that he could produce concussion more readily if he first ligated one or more of the arteries to the brain. He finally concluded that cerebral anemia alone would cause concussion, and that concussion was the result of two factors—cerebral anemia and "ébranlement" or molecular disturbance. Kocher,[11] Kramer,[38] Cannon [32] and others, after experimental investigation, supported the hypothesis that cerebral anemia is the sole cause of concussion.

Fischer,[39] and later Miles,[40] thought that the cerebral anemia was due to reflex contraction of the blood vessels owing to the sudden movements of the cerebrospinal fluid following the blow, which was assumed to stimulate the restiform bodies. In the light of present knowledge concerning the vasomotor supply of cerebral vessels, this theory must be abandoned.

Witkowski [41] produced a condition similar to concussion in the frog after removal of the heart. Cerebral anemia could hardly be a factor under these circumstances. Though the evidence supporting the theory that cerebral anemia is the direct cause of concussion appears to be comparatively conclusive, several investigators, Tilman,[42] Rahm,[43] Bres-

35. Hill, L.: The Cerebral Circulation, London, 1896.

36. Küssmaul and Tenner, quoted by Hill, L.: The Cerebral Circulation, London, 1896.

37. Polis, A.: Rev. de chir. **14**:274 and 645, 1894.

38. Kramer, S. P.: Ann. Surg. **23**:163, 1896.

39. Fischer, H.: Volkmann, Samml. klin. Vortr. se. 1 **27**:219, 1871.

40. Miles, A.: Brain **15**:153, 1892.

41. Witkowski, L.: Virchows Arch. f. path. Anat. **69**:498, 1877.

42. Tilmann: Arch. f. klin. Chir. **59**:236, 1899.

43. Rahm, H.: Zentralbl. f. Chir. **47**:146 (Jan.-June) 1920.

lauer,[44] Duret,[45] Koch and Filehne[23] arrived at very different conclusions.

For instance, the opposite conclusions of Koch and Filehne[23] are most striking. They produced concussion by means of repeated light blows in place of the single severe blows used by most other investigators. Repeated light blows would not alter the conformation of the skull to a degree sufficient to cause cerebral anemia, but might, through summation of effect, cause an effect on the central nervous system similar to that caused by a single severe blow. These investigators concluded that concussion was the result of molecular disturbance or "ébranlement." Bergmann[25] supported these conclusions, and the hypothesis received considerable popularity. By molecular disturbance they evidently meant that concussion was due to a direct mechanical effect on the structural equilibrium of the cells.

Brief mention must be made of Duret's[45] theory of cerebrospinal shock as the cause of concussion. Duret forced fluid into the lateral ventricles to reproduce the sequence of events in the skull following the appearance of the zone of indentation caused by a blow. This lessening of the volume of the skull raises the intracranial pressure so that the cerebrospinal fluid in the ventricles is forced violently through the aqueduct of Sylvius. It then is supposed to impinge on the floor of the fourth ventricle, and to cause the minute contusions and petechial hemorrhages so often found in the region of the aqueduct in persons dying from the results of injury of the head. Duret produced similar lesions in his experiments, and considered these lesions as the pathologic basis of concussion. The paralytic symptoms of concussion were due to reflex anemia, the wave of cerebral spinal fluid stimulating the restiform bodies which caused reflex contraction of the intracranial blood vessels. Miles,[40] after a series of experiments of a similar nature, later supported Duret's work. He considered, further, that the scattered petechial hemorrhages so often found in persons dying of concussion were due to pressure changes on the arterioles caused by this sudden rush of cerebrospinal fluid. This produced rupture of the thin-walled vessels. As opposed to this theory, Tilanus and Deucher[46] were able to produce experimental concussion after aspirating the cerebrospinal fluid. Kocher[11] later demonstrated lesions in a patient who had not suffered a primary loss of consciousness similar to those described by Duret. These experiments, together with the difficulty of considering the results of a blow causing concussion as similar to the increase of intracranial pressure produced by Duret, have led to the abandoning of his hypo-

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44. Breslauer: Beitr. z. klin. Chir. **121**:590, 1921.

45. Duret: Arch. de physiol., 1874, p. 320; quoted by Tilmann: Arch. f. klin. Chir. **59**:239, 1899.

46. Deucher: Deutsche Ztschr. f. Chir. **35**:145.

thesis. That reflex cerebral anemia of any generalized extent does not occur, has already been stated.

The recent work of Breslauer [44] is not well known. Breslauer found that he could not produce unconsciousness by manual pressure on the cerebrum in dogs, for only when the medulla was involved did unconsciousness occur. He concludes that concussion is due to purely medullary effects. All physiologic evidence, however, tends to place the seat of consciousness in the cortex, and mild cases of concussion show scarcely any stimulation or inhibition of the medullary centers, so that it is difficult to accept Breslauer's reasoning.

A different and interesting line of thought was followed by Tilman,[42] who ascribed concussion to the tearing apart of the white and gray matter. As the white and gray substance of the brain have different specific gravities, they attain different degrees of acceleration in the induced field of gravity set up by an impact on the head. The tendency is for them to be torn apart, the resulting solution of continuity causing unconsciousness. The prompt recovery in most cases of concussion makes this hypothesis untenable. Rahm,[43] however, applies this line of reasoning to the cell particles forming nervous tissue. Protoplasm is an emulsion containing, among other things, lipoids, large protein molecules and electrolytes suspended in a watery base. These particles, having different specific gravities, take up new positions relatively in response to the new gravitational field set up by the violent movement of the brain in toto (Felizet) which invariably follows a severe impact. Until these particles resume a normal equilibrium in the earth's gravitational field, the cells cannot function. Thus the generalized paralytic conditions seen in concussion are explained. This is purely hypothetic, but is apparently in accord with physical possibilities. Examination of the active cells, vitally stained, shows the particles of the protoplasm in constant movement. This, however, does not altogether rule out the possibility of Rahm's hypothesis being correct to a certain degree.

Trendelenburg [47] denied that concussion is caused by anemia, suggesting that the unconsciousness is caused by a physiologic loss of function of nerve cells, which corresponds closely to that produced by narcosis. I would suggest, however, that narcosis is invariably due to a chemical agent, usually a lipoid solvent, which apparently reverses the polarity of the synapse (Bayliss [48]) replacing excitation by inhibition. It is difficult to give sound physicochemical reasons for an analogy between narcosis and concussion. The one is a chemical effect on the synapsis first, the other purely mechanical.

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47. Trendelenburg, F.: Deutsches med. Wchnschr. **36**:1, 1910.

48. Bayliss, W. M.: Principles of General Physiology, London, Longmans, Green & Company, p. 427, 1920.

MEDULLARY EFFECTS IN CONCUSSION

The confusion concerning the physiologic and pathologic basis of concussion is thus evident. Careful examination of the experimental methods adopted by past investigators in the study of concussion shows that in most cases a general anesthetic has been used. This obscures the predominant symptom of concussion, namely that of immediate and complete unconsciousness. Further, cats, dogs and rabbits have been used for most experiments. As these animals, belonging to a much lower order in the scale of evolution than man, depend to a much greater degree on spinal reflexes, the results of injury of the head might correspondingly be different. Again, most investigators have carefully recorded the blood pressure, the pulse rate and the respiratory rhythm, noting the changes produced by blows on the head, and have drawn conclusions from them. The vast majority of mild cases, however, show very slight if any medullary effects while the severe cases show an immediate arrest of respiration with stimulation of the vagus and vasomotor centers. In the present investigation many of the experiments of previous workers were repeated, but it soon became evident that little could be gained from such methods. To cause medullary effects, the blows are necessarily sufficiently severe to cause coincident contusion and hemorrhage. The medullary effects produced are those of stimulation or inhibition of the bulbar centers. Thus any agency which will stimulate or inhibit these centers, produces changes similar to those produced by blows on the head. The onset of concussion consequently cannot be judged from changes in the blood pressure, the pulse rate or the respiratory rhythm.

EXPERIMENTAL INVESTIGATION OF MEDULLARY EFFECTS UNDER VARYING CONDITIONS

In the following experiments, the work of several previous investigators was repeated in order to judge the value of blood pressure changes and alterations in the respiration and pulse rate in the demonstration of the physiologic and pathologic changes in varying types of injury of the head. Dogs and cats were used and were anesthetized with either ether or amytal. The respiratory rhythm was recorded through a tube inserted in the trachea, one of the arms of which connected with a writing lever resting on a tambour. The blood pressure was recorded in the usual manner by connecting a cannula placed in the carotid to a mercury manometer, on one arm of which floated the writing pointer. Comparison was made of the medullary effects produced by the following agencies.

1. Blows on the head.
   (*a*) Single severe one.
   (*b*) Repeated light taps.

2. Electrical current on the medulla oblongata.
   (*a*) A weak induction current.
   (*b*) Galvanic current of 120 volts.
3. Direct mechanical pressure on the medulla oblongata.
4. Cerebral anemia.

The striking similarity in the alterations in blood pressure curve and respiratory rhythm produced by these different means readily shows the impossibility of demonstrating the etiology of concussion by these methods.

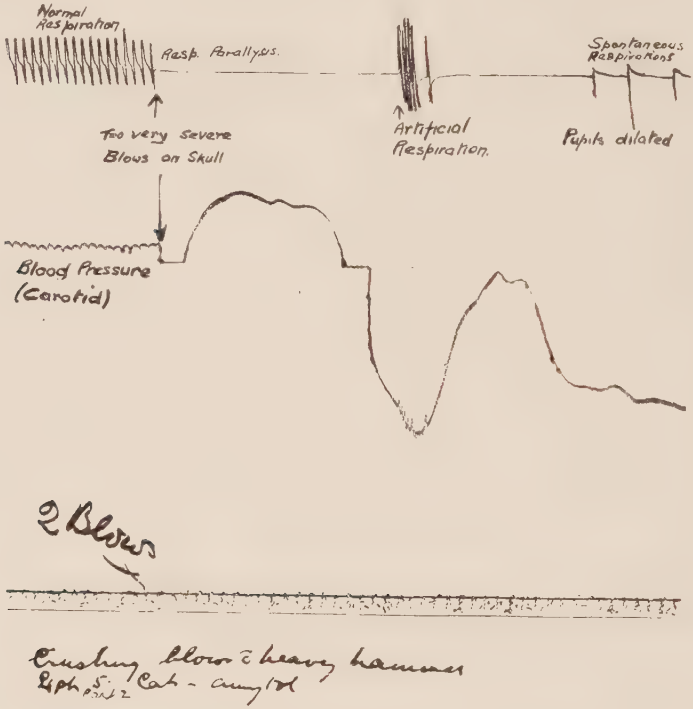

Fig. 1.—Respiratory and blood pressure changes following a severe blow on the head of a cat. Note arrest of respiration with an immediate fall in blood pressure, followed by a rise.

Experiment 1.—*Effect of severe blows on the head.*

A well nourished cat was used for this experiment. The first blow was delivered on the head with a wooden mallet weighing 175 Gm. The blow was moderately severe but did not produce any marked changes in blood pressure, although a slight rise was evident with a deep respiratory effort. Two severe blows were now inflicted on the head of the same animal with a metal mallet weighing 400 Gm. Respiration immediately stopped, with a sudden fall of blood pressure and arrest of the heart action. The blood pressure changes after the recovery of the immediate fall shown in figure 1 are essentially those following arrest of respiration in asphyxia.

Experiment 2.—*Effect of repeated light blows on the head of animals.*

This experiment was based on those of Koch and Filehne. Dogs were used under amytal anesthesia. The repeated light blows, inflicted with a light wooden mallet produced marked stimulation of the bulbar centers as seen in the deep, rapid respirations and rising blood pressure. There was also slowing of the pulse rate, evidently due to stimulation of the vagus center. When these blows were continued over a long period, respiratory paralysis finally occurred. With the arrest of respiration, the pulse rate and blood pressure curve (figs. 3 and 4) followed changes almost similar to those noted in the previous experiment (fig. 1) although there was not the marked initial rise in the blood pressure. This was evidently due to the fact that the vasomotor center had been stimulated by the

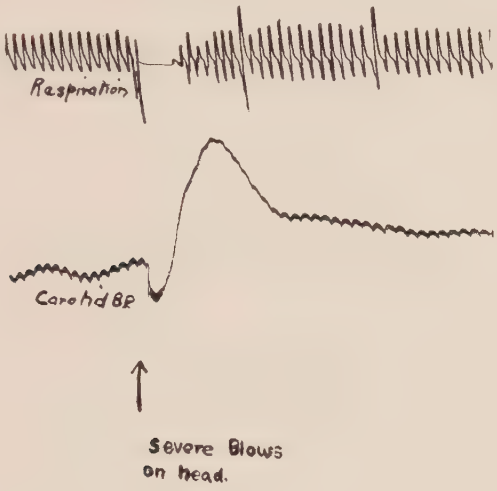

Fig. 2.—Respiratory and blood pressure changes following a severe blow on the head of a cat.

blows from the outset of the experiment, and was not capable of further stimulation by the increasing H-ion concentration resulting from this secondary asphyxiation.

Experiment 3.—*Effect of electrical stimulation of the medulla.*

A dog was used under amytal anesthesia in this experiment. The medulla was exposed by removal of the atlanto-occipital ligament. Stimulation of the medulla with a weak induction current produced marked stimulation of the respiratory center with a fall of blood pressure soon followed by a rise (fig. 5). When a current of 120 volts was allowed to flow through the medulla there was an immediate fall of blood pressure followed by a brief rise which soon fell to zero. Analysis of this curve (figs. 6 and 7) again shows a similarity to that produced in asphyxia.

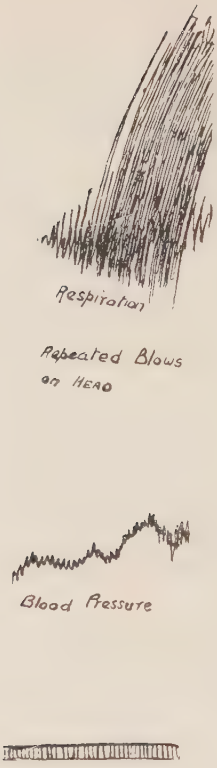

Fig. 3.—Early effect of repeated light blows on the head of a dog. Note the stimulation of the respiratory and vasomotor centers.

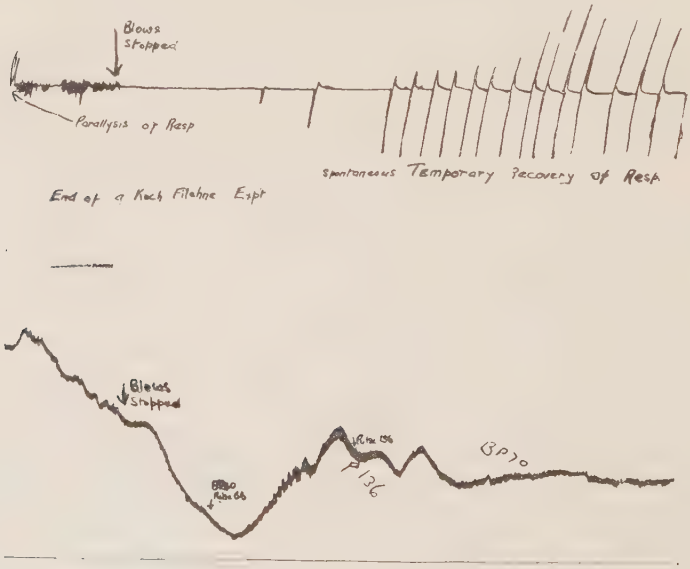

Fig. 4.—End of experiment shown in figure 2. Note that the respiratory center is temporarily paralyzed. The blood pressure rapidly falls, probably due to exhaustion of the vasomotor center.

Experiment 4.—*Effect of weight falling on the dura.* (Modified experiments of Maassland and Saltikoff.)

A large dog was used under ether anesthesia. Two holes were trephined in the skull, one on either side. Into one was screwed, in air-tight fashion, an instrument for recording the rise in intracranial pressure. The rubber membrane over the end of the inner tube rested directly on the brain. The other end of the tube was connected with a piston recorder. A long brass tube was screwed into the other hole, protected from resting on the dura by a brass flange. The dura on this side was not incised. A known weight could thus be allowed to fall down this tube on to the dura from any height, and the effects on the intracranial pressure were noted. When a weight of 350 Gm. was allowed to fall from a height of 6 inches, respiratory and vasomotor stimulation occurred (fig. 8). There was an immediate slowing of the pulse rate due to vagus stimulation. When

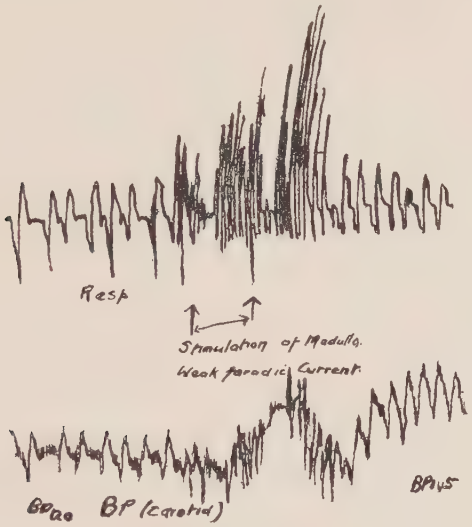

Fig. 5.—Effect of a weak induction current on the medulla. Note the marked stimulation of the bulbar centers.

the weight was allowed to drop from a height of 27 inches, there was immediate cessation of respiration, with a blood pressure curve again similar to that of asphyxia when the blood pressure had recovered from the immediate fall which occurred (fig. 9). The intracranial pressure showed an immediate rise and fall due to the movement of the brain (fig. 10). There soon followed a gradual rise, as shown by Cannon, which he ascribed to the edema resulting from the trauma to the brain tissue, though it may be in part due to resulting small hemorrhages.

Experiment 5.—*Direct mechanical effects on the medullary centers.*

Dogs were used under ether anesthesia. The mechanical effects were produced by injecting saline solution into the cisterna magna. When 10 cc. of physiologic sodium chloride at 37 C. were injected, marked stimulation of the bulbar centers occurred (fig. 11). When 12 cc. of this solution was forcibly injected in a similar manner, immediate paralysis of the respiratory center occurred. There was, first, a slight fall of blood pressure, soon followed by changes similar to those produced in asphxia (fig. 12).

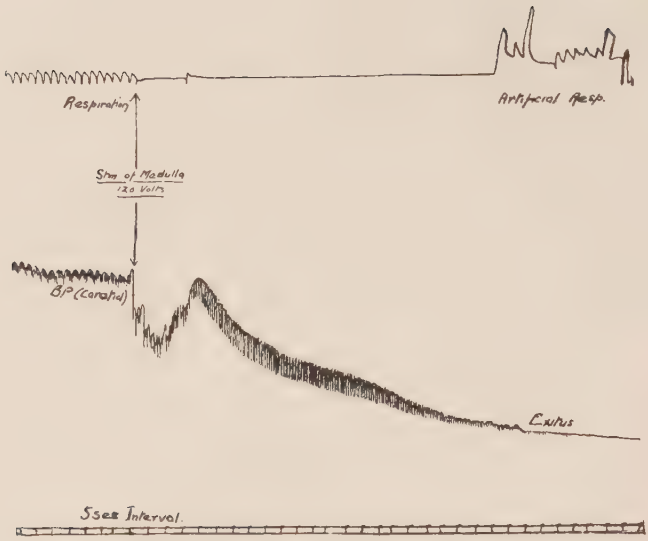

Fig. 6.—Effect of a current of 120 volts on the medulla. Note the immediate paralysis of respiration with stimulation of the vagus and vasomotor centers.

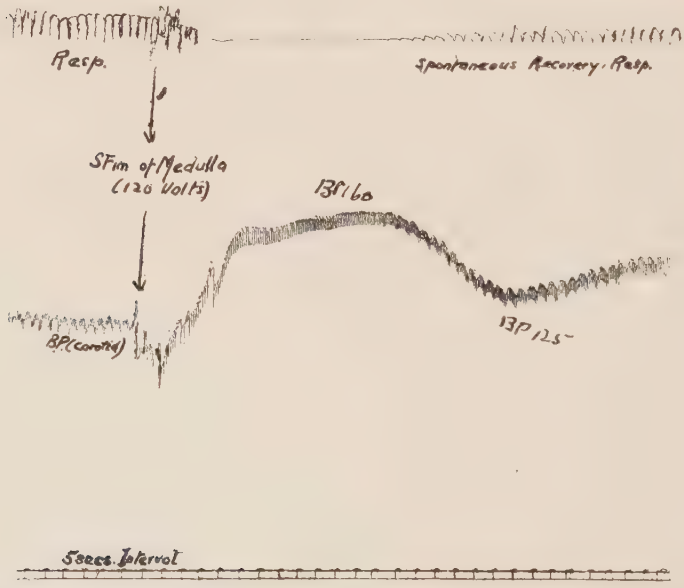

Fig. 7.—Effect of 120 volts of direct current applied to medulla with recovery of respiration.

Fig. 8.—Effect of a weight of 350 Gm. falling on the dura from a height of 6 inches. Note the respiratory and vasomotor centers show stimulation.

Fig. 9.—Effect of a weight of 350 Gm. falling on the dura from a height of 27 inches. Note immediate respiratory paralysis with a fall in blood pressure followed by a sharp rise, then a second slow fall to zero.

The striking similarity of these results is readily apparent if they are compared with similar tracings showing the effects of complete cerebral anemia (fig. 13) and of asphyxiation (fig. 14).

These tracings appear to show that the marked blood pressure changes produced when respiratory arrest is caused by any means are due to asphyxiation. The respiratory center is always the first to be

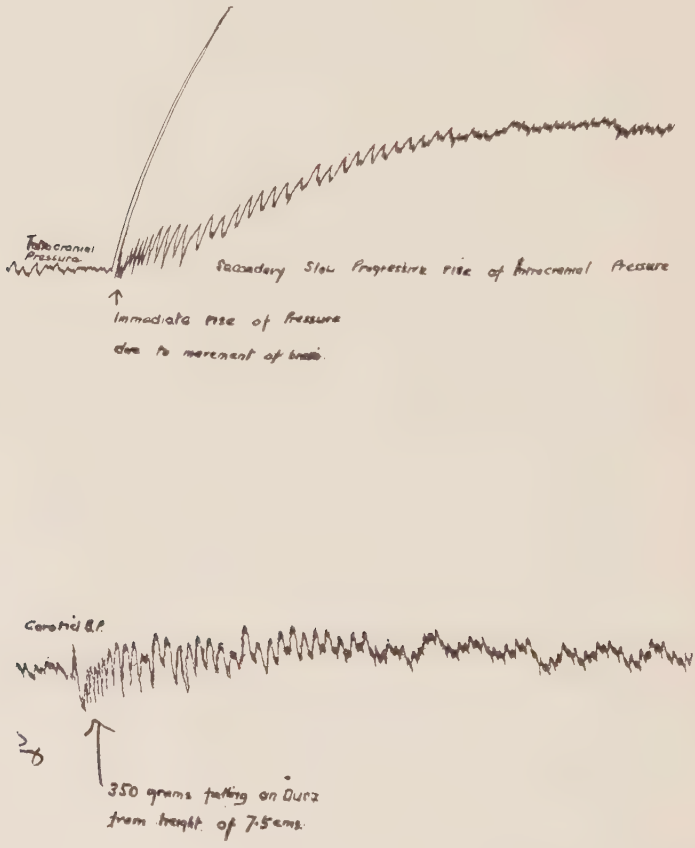
Intracranial Pressure
Secondary Slow Progressive rise of Intracranial Pressure
Immediate rise of Pressure
due to movement of brain
Carotid B.P.
350 grams falling on Dura
from height of 7.5 cms.

Fig. 10.—Effect on the intracranial pressure of 350 Gm. falling on the dura from a height of 3 inches. Note the immediate rise of intracranial pressure due to the movement of the brain, also the gradual rise due to the resulting edema (Cannon [32]).

paralyzed. With mechanical effects not severe enough to paralyze the respiratory center, a marked stimulation of respiratory movements occurs; further, there is an immediate fall of blood pressure, shown by Kocher [11] to be due to vagus stimulation. He pointed out that this fall did not occur when the vagus had been sectioned previously. This sudden fall soon gives way to a rise of blood pressure, due to early

vagus escape when the stimulation of the vasomotor center becomes apparent. The results are the same whether the agency affecting the medulla is mechanical, electrical or nutritional. Cushing has shown that cerebral anemia produces stimulation of the vagus and vasomotor centers. All these agencies stimulate when of moderate degree. Stimulation, however, changes to inhibition, or paralysis, if the external agency is too severe. First, the respiratory center is paralyzed and a

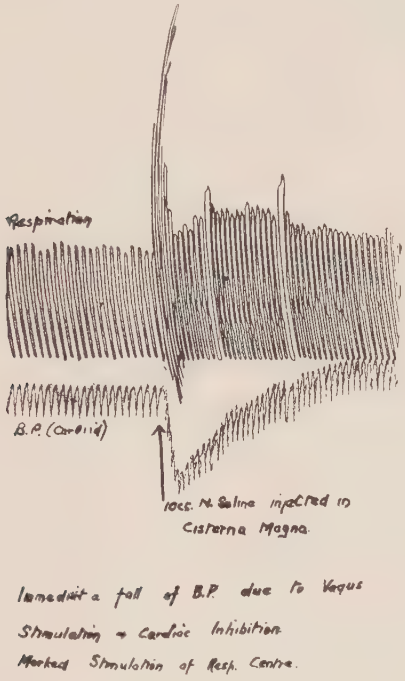
Respiration
B.P. (Carotid)
10cc. N. Saline injected in Cisterna Magna.
Immediate fall of B.P. due to Vagus Stimulation + Cardiac Inhibition
Marked Stimulation of Resp. Centre.

Fig. 11.—Effect of 10 cc. of physiologic sodium chloride injected into the cisterna magna. Note the immediate fall of blood pressure followed by a rise, also the respiratory stimulation.

condition of asphyxia exists. Maassland and Saltikoff were able to paralyze the vagus and vasomotor centers, together with the respiratory center, by placing heavy weights on the dura. This seems possible, though this effect has not been obtained in any of the numerous experiments carried out in the present research. The vasomotor center would appear to have an extraordinary resistance to violence.

The conclusion drawn from these results is that a mechanical effect, such as concussion appears to be, if of a moderate degree, will stimulate the medullary centers, but if of sufficient severity will tend to paralyze them. Von Bergmann has stated that mild degrees of concussion are always accompanied by a slower pulse rate and a rise of blood pressure with a somewhat greater respiratory effort, which is evidently a mechanical stimulation of the centers concerned. Ferrari has shown that in the movement of the brain following an impact on the head there is considerable mechanical violence to the brain. Gussenbauer's case, cited above,

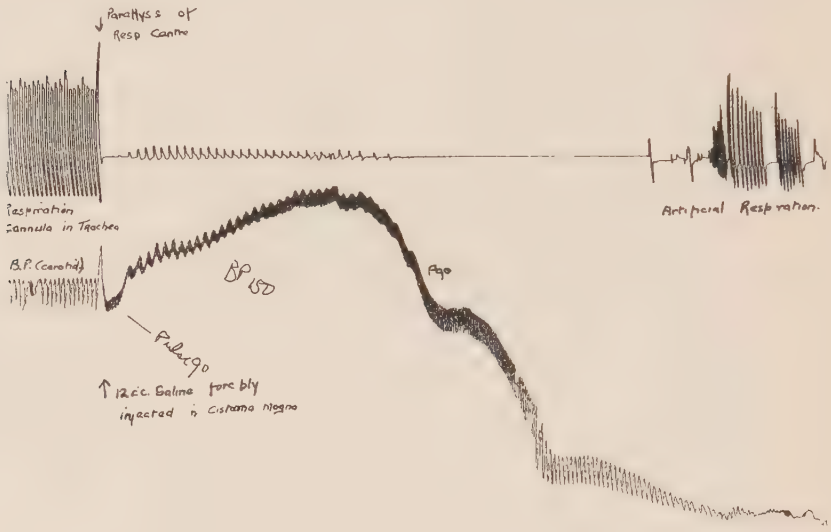

Fig. 12.—Effect of 12 cc. of physiologic sodium chloride forcibly injected into the cisterna magna. Note the similarity of the blood pressure changes to those in asphyxia.

exemplifies this mechanical effect of a sufficient severity to paralyze the respiratory center temporarily. The arrest of the heart was evidently due to vagus stimulation inhibiting the heart action. Thus mechanical violence alone could readily produce the symptoms seen in concussion. Cerebral anemia acts in precisely the same manner; moderate anemia stimulates the medullary centers, while severe anemia paralyzes the respiratory center. Study of the blood pressure and respiratory changes, therefore, will never assist in explaining the place of anemia in the causation of concussion. As there is experimental evidence that anemia

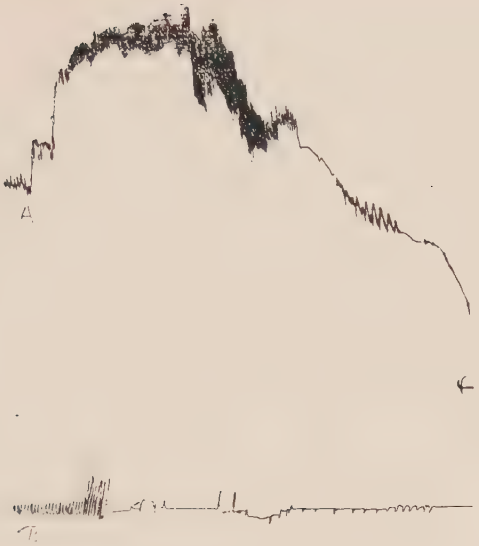

Fig. 13.—The effect of complete cerebral anemia on the respiratory, vasomotor, and vagus centers. (From Hill: Cerebral Circulation.)

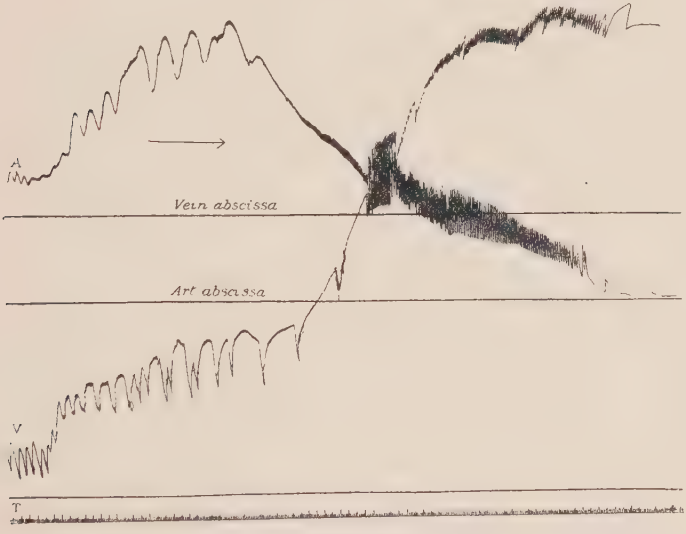

Fig. 14.—Effect of asphyxiation on blood pressure; when the venous pressure curve crosses the arterial pressure curve the pulse pressure increases, and the blood pressure remains constant for a few moments. This also occurs with respiratory paralysis, produced by blows, electricity currents and mechanical pressure. (From Halliburton: Textbook of Physiology.)

may have a place in the causation of concussion (Cannon, Felizet), and as it is generally recognized that cerebral anemia will produce paralytic conditions similar to those occurring in concussion, it is necessary to show conclusively whether sudden anemia will or will not produce immediate unconsciousness. The inbending area of the elastic skull following a blow which forces out the fluids from the skull will immediately spring out again. Any anemia, therefore, must be exceedingly transient, certainly less than one second in duration. It seems improbable that an anemia of such a short duration could cause such widespread paralytic symptoms. All the cells of the central nervous tissue are not adjacent to capillaries, and these cells could hardly be affected by such a transient anemia. Further, it has never been demonstrated, and seems very unlikely, that in the frequent cases of the "knock out" blows the indirect force of the blow on the base of the skull can cause a sufficient diminution in the size of the cranial cavity to squeeze out the fluid contents. It, therefore seems, *a priori,* very unlikely that cerebral anemia is the cause of concussion. The following experiments give considerable support to this contention.

CEREBRAL ANEMIA PROBABLY NOT CAUSE OF CONCUSSION

EXPERIMENT 1.—*Concussion in the heartless frog.*

The heart is removed from a frog under light general anesthesia. On recovery from the anesthesia the frog appears to be conscious. If it receives a sharp blow on the head, a condition arises which appears to be analogous to concussion. The frog lies on its back, without moving and all reflexes are lost. In a few moments it recovers and rights itself and all reflexes return. A blow on the head of the normal frog produces a similar condition analogous to that of concussion in man.

EXPERIMENT 2.—*Complete cerebral anemia in the rabbit without loss of consciousness.*

Under a local anesthetic (procaine hydrochloride), the sternum is opened in the midline, care being taken not to open the pleura. The ascending aorta is dissected out, and clamped for periods of from 1 to 7 seconds. It is evident that this will produce an immediate anemia of much longer duration than that produced in concussion. The animal in this case does not lose consciousness. Immediately on putting it on its feet, it struggles and gives all signs of being conscious. Trapping experience shows that when rabbits are given a sharp blow on the head they immediately lose consciousness for several seconds. Concussion can therefore readily be produced in rabbits by means of blows on the head, but not by transient cerebral anemia of five seconds' duration.

These experiments show that in both the frog and the rabbit, though a condition exactly simulating concussion can be produced readily, it is not the result of cerebral anemia. A transient cerebral anemia even of five seconds' duration does not produce unconsciousness. It is evident, then, that these experiments confirm the arguments stated above, that cerebral anemia is not the cause of concussion. In early experiments

dealing with the effect of immediate cerebral anemia produced by ligating the carotid and vertebral arteries, it is probable that the four arteries were not ligated simultaneously, but that the operation lasted a few seconds. Thus the impression was gained that cerebral anemia causes immediate unconsciousness (Hill [35]).

THE RELATION OF CONCUSSION TO MICROSCOPIC CONTUSION

I have already stated that a great controversy appears in the literature on the question of the relation of concussion to multiple contusions and petechial hemorrhages, many considering the conditions synonymous. If the conclusions reached previously are accepted, it is evident that a mechanical force paralyzing cell function need not necessarily be associated with cell death. It is evident that the generalized, transient, cortical paralysis seen in concussion is not the result of cell death, otherwise prompt recovery could not occur. Following contusion or petechial hemorrhage, an inflammatory reaction and repair process necessarily follow. The immediate symptoms would not, as in concussion, be the most severe, but rather the later symptoms would appear gradually with the development of reactionary edema and further cell death. The lesions would tend to cause a prolonged rise of the intracranial pressure; adjacent tissues would suffer from the edema, and so a protracted course with increasing severity of symptoms might be expected; further, destroyed neurons would not be replaced, but removed, and the lesion would be repaired with fibrous connective tissue. Thus sequelae of a more or less permanent character would result, unless the contusions were restricted to the silent areas. But prompt and complete recovery is generally accepted as one of the outstanding characteristics of concussion. Concussion and contusion can hardly be synonymous, as stated by Kocher.

Concussion can cause temporary paralysis of the respiratory center with prompt recovery. There could be no recovery if this damage were due to a contusion of the respiratory center. Contusion is synonymous with cell death. If the temporary paralysis of the respiratory center in concussion is prolonged beyond narrow limits death will occur. Thus death can occur in pure concussion. This explains why Kramer and Horsley,[38] and later Cannon,[32] by means of artificial respiration, resuscitated animals in which the respiratory center had been paralyzed by blows on the head.

Death, however, rarely occurs in cases of concussion without the added injury of numerous contusions and petechial hemorrhages. The medulla is well protected from injury by its position and only severe violence will cause immediate, prolonged, paralysis of the respiratory center. Ferrari showed how readily contusion of the cerebrum resulted from violence to the head. In response to the blow, the soft brain is

thrown against the bony covering on the opposite side (contre coup). With the narrowing of the diameter of the skull caused by the zone of indentation, the brain may be nipped between the two sides of the cranium. It is possible that, as Duret suggested, the spinal fluid is forced down the narrow aqueduct of Sylvius, causing the lesions so frequently found in patients dying from injuries of the head. Thus it is that cases of injury of the head rarely come to autopsy without showing some gross lesion of the brain. Theoretically, however, concussion alone can kill, when death due to respiratory paralysis will be practically immediate, that is, will occur during the first ten minutes. Deaths occurring after the lapse of some hours are due to contusion, hemorrhages or the results accruing from these, such as edema.

Experimentally, however, it has never been demonstrated that concussion can be produced in animals without the accompaniment of gross lesion. The following experiments were devised in order to support the arguments advanced above. Trypan blue injected intravenously stains all the tissues, except those of the central nervous tissue, a deep blue. Should there be any inflammatory reaction of the central nervous tissue, or any gross lesion, that area will also take on the deep blue stain. This would appear a useful method of showing distinctly whether concussion can be produced without the presence of a gross lesion.

Experiment 1.—Two rabbits were used for this experiment. The first one was given general ether anesthesia. After trephining the skull, a fine needle was inserted into the cortex, the operation being conducted with aseptic surgical technic. A normal rabbit was then given a blow on the head of sufficient severity to cause unconsciousness of short duration. The blow was dealt with an instrument made by filling a thick rubber tube with lead shot, as such an instrument would be less likely to cause hemorrhages and contusions. The unconsciousness was presumed to be a condition analogous to concussion in man. In rabbits recovery from concussion is rapid. Within a minute the animals move about naturally and take food just as before. On each of the following three days, 20 cc. of a 1 per cent solution of trypan blue in physiologic sodium chloride was injected intravenously. On the fifth day the rabbits were killed and the brains hardened in situ by the injection of 50 cc. of a 10 per cent formaldehyde solution into the carotid artery. After forty-eight hours, the brains were removed and examined. In the rabbit in which the needle had pierced the cortex, a deep blue area in the cortex showed clearly where the needle had entered. This was surrounded by an area of pale blue to which, apparently, the inflammatory reaction had extended. The brain of the animal which had received the blow on the head, was without the stained area, neither were there any hemorrhages present, either subdural or scattered through the brain.

These experiments were repeated several times. Wherever the brain was damaged, the damaged area took up the stain and was colored a deep blue. The brains of animals which had suffered from concussion did not take the stain. Some of these animals showed subdural hemorrhages,

but they were not constant in position or degree and could not have been influential in the onset of the generalized cortical paralysis seen during concussion. These experiments demonstrate that concussion and contusion are not synonymous.

COMMENT

It was stated at the beginning of this article that it would be limited to an endeavor to establish the fundamental physiologic and pathologic basis of concussion. The main factors of confusion appear to lie in the difficulty of clearly differentiating concussion from lesions involving definite histologic and anatomic changes. Without general agreement or a definition of concussion, its scope and etiology, further work would appear futile. It has been shown, both by argument and experiment, that the term concussion should be used to denote the generalized, paralytic effects of an impact on the cortex and medulla, and should be distinguished from the term contusion, whether a gross or microscopic contusion is meant, and also from the term hemorrhage, whether reference is made to a diffuse or petechial hemorrhage. On the other hand, the term contusion should be limited to designate those cases in which destruction of cells can be assumed, and in which there is a tendency toward prolongation or aggravation of symptoms. It has been shown further that mechanical force, a stimulant when of small degree, can act like any other external factor, and paralyze cell function if of sufficient severity. The widely accepted hypothesis that cerebral anemia is the cause of cerebral concussion has been disproved by experiment and by argument. If the blood pressure tracing of complete cerebral anemia is compared with that of cessation of respiration by blows, it is seen that in the former the blood pressure rises immediately while the respiratory center shows deep respirations for seven or eight breaths (fig. 11). The paralysis of respiration due to a severe blow on the head is instantaneous, and there is a sharp drop in blood pressure due to vagus arrest of the heart (fig. 1). This evidence alone would cast doubt on the suggestion that concussion is due solely to cerebral anemia. The effects of concussion are immediate, while the paralyzing effects of cerebral anemia are not immediate but develop after five or more seconds. Thus it appears that concussion is the direct result of a sudden mechanical force on the central nervous tissue, which perhaps is the same conclusion at which Koch and Filehne arrived when they suggested that concussion was due to molecular disturbances or "ébranlement."

To endeavor to understand by what mechanism mechanical force can paralyze cell function, is to enter the realm of speculation. It is known that an electrical current which can produce a similar result, disturbs the equilibrium of the charged particles. The positively charged ions, for example, will be attracted toward the negative pole. The effect of a

mild current is stimulating, that of severe ones, paralytic. A mechanical force, suddenly changing the pressures about the cells, may also disturb the equilibrium. The activity of cells depends on the equilibrium of the physicochemical state of the cells and of their environment.

The sudden violence with its rapid increase of pressure and quickly changing gravitational field might conceivably disturb this equilibrium and so cause temporary loss of function. Rahm has suggested that in response to the new gravitational field, the equilibrium of particles of different specific gravity will be disturbed. With this temporary disturbance of the physicochemical and gravitational equilibrium a loss of cell function might occur which, being generalized over the whole cerebrum, would cause the transient unconsciousness so typical of concussion. The medulla, being well protected by its position, would be saved from the greater part of this disturbance, and so paralysis of the medullary centers would be rare, as is actually the case.

Thus concussion readily lends itself to an etiologic explanation on purely mechanical grounds. Cerebral anemia is not involved. Contusions, though also mechanically produced, represent a more advanced lesion in that actual cell death is caused. Thus concussion is an immediate, transient, loss of consciousness with or without cell death, caused by the direct action of mechanical violence on the cells of the brain, and with no direct causative relations to cerebral anemia. It is distinct and separate from contusion or hemorrhages, whether minute or large and diffuse.

CONCLUSIONS

1. Concussion is immediate in onset, and tends to spontaneous recovery without sequelae.

2. Concussion must be sharply differentiated from all gross lesions, such as contusion and multiple petechial hemorrhages.

3. The most important symptom of concussion is complete unconsciousness with or without medullary symptoms.

4. Cerebral anemia is not a factor in the causation of concussion.

5. Concussion appears to be due to direct mechanical action on the cells which causes a disturbance of cell equilibrium and temporary loss of function.

6. The medullary effects are those of stimulation or paralysis of the respiratory, vagus and vasomotor centers.

7. The respiratory center is the first to be paralyzed. As this is usually a temporary paralysis, artificial respiration may save life.

8. Death from concussion is immediate and is due to respiratory paralysis with consequent asphyxia.

For help and advice in the preparation of this paper I wish gratefully to record my sincere thanks to Dr. Edward Archibald.

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